Case Summary

"Panda"
Havanese. Male, born October 11, 2002
Black and white. Microchip 135112117A

Panda had a normal, uneventful medical history as a young dog. He had several, relatively mild digestive upsets in his first three years of life but they were always short term and responded to conservative treatment.

In February 2006, Panda presented with a history of vomiting, lethargy, increased thirst and urination of several days duration. Blood testing revealed a significant elevation of liver enzymes. These were the first blood tests performed on Panda. The medical symptoms responded well to medical treatment but follow up blood tests performed over the next several months revealed a persistent and significant elevation of liver enzymes. In April, 2006 we referred Panda for ultrasound examination and possible liver biopsy. The liver was found to be small in size and attempts to obtain a percutaneous liver biopsy were unsuccessful. Because Panda was doing very well clinically we elected not to do a more invasive laparotomy to further examine and biopsy the liver.

Panda gained weight and was neutered in July 2006. Periodic blood tests showed fluctuating but persistently elevated liver enzymes. Occasional episodes of anorexia, lethargy, &/or vomiting, responded to fasting and short courses of medication (metronidazole and amoxicillin).

In June, 2008, Panda presented with significant abdominal distension caused by fluid accumulation (ascites). Laboratory tests indicated ongoing presence of liver disease/inflammation and a depressed level of serum protein. Fluid analysis of the abdominal fluid showed it to be a transudate consistent with low serum protein levels. This finding would be consistent with progressive liver disease and decreased production of protein by the liver. No other abnormalities were revealed by the blood chemistry. In mid July 2008 we repeated ultrasound exam of the abdomen. Findings were; decreased liver size, enlarged gall bladder, increased pancreatic size and thickened and irregular densities within the mesentery. Some concern was felt about possible neoplasia in addition to progressive liver disease.

Paliative care for Panda continued through July and August and consisted of drainage of abdominal fluid. Up to 1 litre of fluid was removed at each treatment and helped Panda function at a remarkably high level until late August, 2008. At that time due to progressive weight loss and increasing impairment of respirations caused by abdominal fluid, the decision was made to euthanize Panda.

An autopsy was performed and revealed abnormalities in the following areas:
Liver- abnormally small, light brown color and nodular appearance.
Gall Bladder- at least double normal size, thickened wall and thickened sludge like contents.
Kidneys-Several scarred areas indicating old infarcts.
Other organs appeared normal on examination. Of note is that no sign of neoplasia was found at the autopsy.

Sections of liver, kidney and gall bladder were sent to Dr. Brian Wilcock for histopathological examination. His report is enclosed in it's entirety.

Summary

The histopathology report describes two distinct disease conditions that were present in Panda at the time of death.

The presence of kidney disease was unexpected because there had been nothing in the lab tests or clinical signs that had indicated it's presence. The hypoproteinemia that had caused the ascites would have been exacerbated by protein loss in the urine. In this respect it would have accelerated the deteriorating condition. As more time went by, the kidney disease would have become more significant, because this type of condition typically has a progressive nature.

The liver was the site of the most significant pathology. The absence of the normal structural components and an abnormal vascular pattern means that this liver had never been capable of functioning in a normal manner. This is not an acquired condition, it would have been present since birth and as such would be described as congenital defect.

Conversation with Dr. Wilcock leaves some question as to whether this can be classified as a classic microvascular dysplasia, but clearly, there is a profound disruption of the normal liver anatomy which would have a significant negative impact on the function the organ. Another anticipated consequence of these vascular anomalies would be an alteration of normal blood pressure dynamics in the abdomen. This in turn would contribute towards the severe ascites that Panda experienced in the last months of his life.

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DATE RECEIVED: 9/12/2008
DATE REPORTED: 9/13/2008

CASE NUMBER: H15463-08

OWNER: Johnston
PATIENT: "Panda"

BREED: Havanese
SEX: M

AGE: 6 yrs.
TISSUES: Liver & kidney

I started with kidney, where there is an unexpected lesion of diffuse glomerular thickening because every capillary loop is diffusely hyalinized. There is hypertrophy of the Bowman's epithelium and, in many cases, there are multiple adhesions of the second glomerular tuft to that Bowman's membrane. In contrast to the glomerular disease, the tubules actually look fairly good. There are too many tubules that are basophilic and flattened, indicating at least some degree of ongoing ischemic tubular injury. As the glomeruli become progressively thickened, they impair the effective delivery of blood from the efferent arteriole to these proximal tubules. The onset of ischemic tubular disease marks the transition from subclinical protein-losing nephropathy into actual uremic renal failure.

There is a second kidney lesion corresponding to your macroscopic observation of wedges of cortical scarring. Those wedges are characterized by virtually complete loss of tubules, replacing those tubules with condensed fibrous stroma and sclerotic glomeruli. Because virtually all of the tubules have disappeared, those glomeruli are now situated one right against the other. This is a very old lesion and it likely has no functional significance when contrasted to the glomerular disease that is affecting every single glomerulus in both kidneys. This is unrelated to the glomerular disease.

The gallbladder looks histologically normal, but the liver is a disaster. That liver has an unexpected lesion characterized by a great deal of sinusoidal vascular dilation that creates what looks like a whole series of blood-filled cysts throughout the liver. In contrast to what I was expecting, there is very little fibrosis and I am having trouble detecting traditional changes of cirrhosis or chronic inflammation. The hepatocytes themselves seem to be arranged in jumbled clusters, and it is often difficult to identify orderly hepatic cords. The portal triads have an irregular and sometimes seemingly random distribution, surrounded by pigment-laden macrophages but no other leukocytes.

I find it very difficult to judge the functional significance of these lesions. I do not see hepatocellular necrosis, but surely all of these dilated sinusoids mean that blood is not traversing this liver as it should. I could easily imagine this lesion giving rise to portal hypertension and ascites, with that ascites perhaps made even worse by the presence of protein-losing nephropathy. What I do not know is the pathogenesis for this lesion, and I am slightly hampered by not knowing the age of this dog. That missing information impacts my ability to interpret both the kidney lesion and the liver lesions. Theoretically, this liver could represent rare congenital microvascular dysplasia.

DIAGNOSIS:

1. IN KIDNEY, DIFFUSE GLOMERULONEPHRITIS WITH MILD ISCHEMIC TUBULAR INJURY AND ONGOING REPAIR, SIGNIFICANT AS A CAUSE FOR PROTEIN-LOSING NEPHROPATHY AND, PROBABLY, MORE RECENT UREMIA
2. IN LIVER, A BIZARRE LESION OF WIDESPREAD SINUSOIDAL VASCULAR ECTASIA AND SUBTLE HEPATIC ARCHITECTURAL DISORGANIZATION, PROBABLY SIGNIFICANT AS A CAUSE FOR PORTAL HYPERTENSION BUT I DO NOT KNOW THE PATHOGENESIS FOR THIS STRANGE LESION (NOT INFLAMMATORY, NOT TOXIC, AND CERTAINLY NOT NEOPLASTIC)

BRIAN P. WILCOCK, D.V.M., Ph.D., VETERINARY PATHOLOGIST

Questions about this report? Call or FAX me at 1-800-853-PATH